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Title

Should I stop or should I grow? Light, ethylene and PIF3 team up to favour the appropriate response.

Authors

Séverine Lorrain and Christian Fankhauser

Centre for Integrative Genomics, Faculty of Biology and Medicine, University of Lausanne, CH-1015 Lausanne, Switzerland. christian.fankhauser@unil.ch, severine.lorrain@unil.ch

Short summary

Plant growth is tightly controlled through the integration of environmental cues with the physiological status of the seedling. A recent study now proposes a model explaining how the plant hormone ethylene triggers an opposite growth response depending on the light environment.

Being sessile, plants adapt to their surrounding environment by changing their shape and their development. Different environmental cues such as light quantity, quality or temperature are integrated with the physiological and hormonal status of the plant to trigger appropriate organ and tissue-specific responses [1]. The embryonic stem (hypocotyl) of *Arabidopsis thaliana* is a good model to study the crosstalk between environment and hormones in the control of growth [2, 3]. When seed germination occurs in darkness (in the soil), the hypocotyl quickly elongates to reach the light to allow the seedling to start its photoautotrophic life style (de-etiolation) [4]. In direct sunlight hypocotyl growth slows down and presents a rhythmic pattern controlled by the circadian clock [3]. Increased temperature or changes in the light quality indicative of the presence of neighbour plants also modulate the rate of hypocotyl elongation in de-etiolated seedlings [5-9].

Hypocotyl elongation depends on the interplay between at least 4 different classes of hormone: auxin, gibberellins, brassinosteroids and ethylene [2, 3]. They each have their

own perception and signalling pathways, in addition these pathways influence each other at different levels. Furthermore sensitivity to one hormone depends on the physiological status of the seedlings. This internal/hormonal status of the seedling is influenced by the environment explaining why the effect of hormone application depends on the surrounding environment. For instance auxin-induced hypocotyl elongation presents a typical bell-shaped dose response that is modulated by the light intensity [7, 10]. Recent work by Zhang et al revisits another environment-dependent hormone response: the influence of light on the effect of ethylene on hypocotyl elongation [11].

Ethylene is a gaseous hormone well known for its effect on fruit ripening that also affects numerous aspects of plant development [12]. Treatment with an ethylene precursor triggers two opposite responses on Arabidopsis hypocotyls with inhibition of elongation in the dark and promotion of elongation in the light. Zhang et al now show that the ethylene-induced hypocotyl elongation requires not only the presence of light but also a certain quantity of light since in low fluence rates or in days with less than 8 hours of light, ethylene treatments inhibit hypocotyl elongation [11].

In the search for proteins involved in this response, Zhang *et al* tested whether the usual suspects linking light and hypocotyl elongation, the PIFs (Phytochrome Interacting Factor), were involved {Lau, 2010 #20}{Leivar, 2011 #7}. These proteins are bHLH transcription factors that interact with red/far red photoreceptors called the Phytochromes [13]. In red-rich environments typical of direct sunlight the active phytochromes inhibit the PIFs through phosphorylation and/or degradation, leading to reduced hypocotyl elongation [13]. However in conditions where phytochromes are inactive such as in darkness or in far-red rich environments (foliar shade), PIF proteins accumulate and promote hypocotyl growth. Interestingly depending on the stimulus PIFs either play redundant, additive or specific roles. For instance PIF1, 3, 4 and 5 are all required to promote full hypocotyl elongation in the dark [14, 15]. Proximity of neighbour plants is detected through changes in light quality (red/far-red ratio) and triggers hypocotyl elongation that is primarily dependent on PIF7 [9]. On the other hand promotion of hypocotyl elongation in response to a reduction of photosynthetically active radiation (PAR) indicative of direct shading mainly involves PIF4 and PIF5 [7, 16].

Finally enhanced hypocotyl growth triggered by an increase in temperature is mediated by PIF4 in light-grown plants [5, 6].

Interestingly in constant light ethylene-induced hypocotyl elongation specifically requires PIF3 and not PIF1, 4 or 5 [11]. Ethylene perception directly activates *PIF3* expression through binding of an ethylene-responsive transcription factor called EIN3 on the *PIF3* promoter. While PIF3 is required for ethylene-induced hypocotyl elongation in the light it is not involved in the ethylene response in darkness, when hypocotyl elongation is inhibited. This cannot be explained by the redundant/additive activity of PIFs in darkness since ethylene still inhibits hypocotyl growth in a mutant lacking PIF1, 3, 4 and 5 [11]. On the other hand a previous study has shown that over-expression of PIF5 leads to an overproduction of ethylene and reduced hypocotyl elongation specifically in darkness [17]. The crosstalk between PIFs and ethylene in the environmental control of growth may thus be more complex.

The next question is how a hormone triggers an opposite response in the same organ depending on the light environment. It appears that this is controlled by the light-environment and not by the hormone itself as ethylene triggers *PIF3* expression in the dark as well as in the light [11]. However PIF3 protein accumulation is light controlled with the protein being more stable in the dark than in the light. Thus changes in PIF3 protein accumulation due to increased gene expression have a more pronounced effect (in relative terms) in the light. However this does not explain why ethylene perception inhibits hypocotyl elongation in darkness. Zhang et al showed that ERF1, another target of EIN3, inhibits hypocotyl elongation and thus the activities of ERF1 and PIF3 antagonize each other [11]. *ERF1* expression is induced by ethylene but the protein is stabilised in the light and destabilized in darkness. Furthermore *ERF1* over expression inhibits hypocotyl elongation in darkness as well as in the light. Thus the PIF3 and the ERF1 pathways are both activated by ethylene but depending on the light environment, one or the other dominates the growth response. The balance of these activities that is influenced by light ultimately determines the effect of ethylene on hypocotyl elongation [11].

As in many studies, these results raise new questions such as whether the ERF1 and the PIF3 pathways interact and if so how? Furthermore, if PIF3 is a major component in ethylene-mediated hypocotyl growth, which pathways are downstream of PIF3? Does PIF3 control the biosynthesis, transport or signalling of the growth-promoting hormone auxin as it has been recently shown for PIF4, 5 and 7 [7, 9, 10, 18, 19]? The role of PIF3 in ethylene-mediated hypocotyl growth was analyzed by artificially increasing the ethylene production, how does this data relate to what happens in normal conditions with physiological level of ethylene? One possibility would be that in darkness when the soil is compact the seedlings produce the stress hormone ethylene leading to a thickening (and reduced lengthening) of the hypocotyl that may be required to grow through the soil [12]. In the light ethylene production has been shown to occur in shaded environments [20], this hormone production may contribute to the elongation response typical in shaded plants by triggering *PIF3* expression that is required for full shade-induced growth [8].

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